



Original Article

Sleep fragmentation and sleep-disordered breathing in individuals living close to main roads: results from a population-based study



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ARTICLE INFO

Article history:

Received 3 June 2013

Received in revised form 30 October 2013

Accepted 31 October 2013

Available online 10 January 2014

Keywords:

Traffic noise

Sleep fragmentation

Sleep-disordered breathing

Heart rate variability

Gender

Cohort studies

ABSTRACT

Background: Nighttime traffic noise is associated with sleep disturbances, but sleep fragmentation and sleep-disordered breathing (SDB) have not been demonstrated in individuals living near busy roads.

Methods: We asked 1383 participants to answer a health questionnaire and to undergo 24-h electrocardiogram (ECG). Nocturnal ECG records were used to calculate the very low frequency index (VLFI) interval, a surrogate marker of sleep fragmentation. Distances of participants' addresses to roadways were calculated using the VECTOR25© Swisstopo roads classification, a traffic noise proxy. Distances of homes within 100 or 50 m of major roads defined proximity to busy roads. Adjusted multivariate logistic regressions analyzed associations between the distance of home to main roads and VLFI or self-reported SDB. **Results:** Distance of participants' homes to main roads was significantly associated with the VLFI in women (odds ratio [OR], 1.58 [confidence interval [CI], 1.03–2.42]; $P = .038$) but not in men (OR, 1.35 [CI, 0.77–2.35]; $P = .295$). Women under hormonal replacement therapy (HRT) were at higher risk for increased VLFI when living close to main roads (OR, 2.10 [CI, 1.20–3.68]; $P = .01$) than untreated women ($P = .584$). Associations with self-reported SDB were not statistically relevant.

Conclusions: In our large population, women living close to main roads were at significantly higher risk for sleep fragmentation than men. The 2-fold higher risk for menopausal women under HRT underscores the vulnerability of this group.

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1. Introduction

Ambient noise is a well-established cause of nuisance. A large body of evidence gathered in the recent years consistently shows that persistent exposure to noise adversely impacts health, and its detrimental effects can vary from subjective symptoms (e.g., tiredness, irritability) to the development of systemic disease (e.g., hypertension, cardiovascular morbidity) and decrease in quality of life [1–6].

Road traffic during the night is a main source of noise affecting the quality of sleep in exposed individuals [7]. Changes in the sleep architecture induced by noise often have been reported to modify sleep stages, induce frequent arousals, and increase the duration of

nocturnal awakenings. Sleep fragmentation reduces the total time of effective sleep resulting in chronic sleep deprivation, a disorder which frequently has been associated with daytime fatigue and decreased neurocognitive capacity [8,9]. Early experimental studies have shown that noise-induced sleep fragmentation impacts on daytime alertness [10]. In addition, it increases upper airway collapsibility [11]. Increased upper airway resistance and inspiratory efforts during sleep may lead to multiple arousals and are associated with sleep-disordered breathing (SDB), a large spectrum condition including patients with regular snoring to those with more severe disease such as those affected by obstructive sleep apnea-hypopnea syndrome (OSAS) [12]. Recurrent sleep fragmentation and obstruction of the upper airways leading to transitory oxygen desaturation and nocturnal arrhythmias frequently are present in patients with SDB [13].

The cardiovascular effects of exposure to traffic noise have been widely documented [3,14–17]. Traffic noise during sleep was

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related to autonomic arousals and increased heart rate [18]. Moreover, altered nocturnal heart rate variability (HRV) has been reported in individuals with SDB [19,20]. Analyzing the frequency domain of the 24-h electrocardiogram (ECG), Roche et al. [21] showed that the very low frequency segment of the spectrum reflects sympathetic activity, thus offering a relatively simple tool to assess the cardiac autonomic function. Further clinical studies by the same group have indicated that the very low frequency index (VLFI) derived from the frequency domain of the 24-h ECG is a surrogate marker of sleep fragmentation, notably when its value exceeds 4% [22,23]. In addition, the VLFI has been validated in a population of elderly individuals [24]. However, the potential link between living in close proximity to busy roads and the VLFI marker of sleep fragmentation has not been investigated.

The SAPALDIA (Swiss Study of Air Pollution and Health in Adults) cohort study has collected data on several health parameters including a 24-h ECG. The study also has maintained a long-term registry of participants' home addresses, which permits the calculation of the distance of individuals' dwellings to main roads and indirectly estimate the potential exposure to traffic noise during the nighttime. Therefore, the SAPALDIA database offered a unique opportunity to investigate the impact of living close to a main road on sleep fragmentation and self-reported SDB in a large population-based sample.

2. Methods

2.1. Design and study population

SAPALDIA is an adult population-based cohort study initiated in 1991 (SAPALDIA 1) and was designed to investigate the effects of air pollution on the respiratory and cardiovascular health of the Swiss population. The cohort started with 9651 participants randomly selected from the population registry including eight representative areas of the climatic, geographic, and cultural diversity of Switzerland. A follow-up survey (SAPALDIA 2) was conducted 11 years later with 8047 participants. Details on the study and the health assessments have been previously published [25,26]. Briefly during a 2-h visit to the SAPALDIA centers, participants answered a comprehensive standardized online questionnaire administered by a trained fieldworker. The questionnaire assessed, among other aspects, the general health status, risk factors (e.g., smoking, regular alcohol intake), systemic diseases, sleep-related disorders (e.g., OSAS), and sleep habits (e.g., keeping windows open in bedroom during the night), as well as perception of traffic density at home vicinity and noise annoyance. At the same occasion, a 24-h ECG holter monitor was conducted.

Our study included 1721 participants of the SAPALDIA 2 cohort (ages, ≥ 50 years), who answered the health questionnaire and underwent a 24-h ECG recording. After excluding participants reporting cardiovascular disease ($n = 91$), treatment of OSAS ($n = 14$), or regular use of β blockers ($n = 233$), the analyses comprised 1383 participants. Written informed consent was obtained from all study participants. The study was approved by the Swiss Academy of Medical Sciences and by the ethics committees of the regional study sites.

2.2. Assessment of distance of participants' homes to main roads

Participants' addresses recorded between SAPALDIA 1 and 2 were geocoded by matching the addresses to the building registry of the Swiss Federal Statistical Office. The length of street segments within a 200-m perimeter around the home coordinates of the study participants was determined using the VECTOR25© Swisstopo (Swiss Federal Office of Topography) classification [27].

Geographic information data obtained on participants' home addresses were used to determine the distance of individuals' homes to the upper three classes of roadways (freeway, highway, and cantonal roads) out of five different main classes. Consistent with previous work by our group, categorical variables were built combining these three upper classes of roadways further using critical distances of 100 m or 50 m of a main road. The distance within 100 m or 50 m of individuals' home address coordinated to the closest three classes of roadways identified by VECTOR25© was used to define proximity to a main road [28]. Details on the SAPALDIA assignment of individuals based on participants' home addresses were published before in the context of air pollution [29].

2.3. 24-h ECG monitoring and %VLFI measurement from HRV analysis

Participants who agreed to a 24-h ECG measurement received detailed instructions from trained fieldworkers who also placed the electrodes and started the ECG recording. The ECG recordings were performed using digital devices (Aria, Del Mar Medical Systems, Irvine, CA, USA) having a frequency response of 0.5–40 Hz and a resolution of 128 samples per second. Three leads were recorded: a V_1 , a modified V_3 with the electrode placed on the intersection between the left midclavicular line and the lowest rib, and a modified V_5 with the electrode placed on the intersection of the left anterior axillary line and the lowest rib. Participants were asked to continue daily routine activities and to document their medication intake and daily activities by completing a standardized time-activity diary handed by the fieldworker, during the whole recording period. Overall, the mean duration of holter recordings was 22.3 ± 2.1 h. Full details on the HRV variables measurements and their interpretation have been previously published [30]. For the purpose of our study, only holter-recorded tracings comprised between approximately 10:00 pm and 6:00 am were considered for analyses. The average duration of sleep was estimated at 8.1 ± 1.0 h considering the individual's steady decrease of the heart frequency in the beginning of the night period and its increase in early morning as a proxy indicator of sleep status, also assuming that our participants were mostly night sleepers. HRV analyses were conducted by independent scorers blinded to other results obtained from participants included in the study.

Details on the calculation of the HRV domain variables were previously published [22]. Briefly, the HRV analysis was based on validated QRS segments and the calculation of the length of RR intervals. Only regular frequency beats and normal-to-normal beats were considered for analyses. Power spectral analysis of the interbeat interval increment was used to identify the very low frequency oscillations, which are mainly related to sympathetic activation. The frequency-domain component of the 24-h ECG was considered for analysis and the very low frequency power (0.00–0.04 Hz) was calculated. The VLFI generally is expressed as the percent ratio between the power spectral in the very low range over the total power spectral density; in other words, it corresponds to the percent increment interval calculated over the total power spectral density.

2.4. Variables of interest and covariates

The independent variable was the distance of cohort participants' addresses to a main road, as described above. The two main outcomes were VLFI as measured from the 24-h ECG and self-reported SDB using information provided by the health questionnaire. Self-reported SDB was defined by a composite variable combining participants' positive answers to questions addressing respiratory pauses during sleep identified by the partner or diagnosed by the doctor as untreated OSAS. In accordance with

previous studies, VLFI $\geq 4\%$ was considered as indicative of sleep fragmentation [22,23].

The following variables were considered as potential confounders: age, sex, level of education, body mass index (BMI), smoking, alcohol intake, hearing deficiency, comorbidities (e.g., diabetes mellitus, arterial hypertension, chronic respiratory diseases, arthritis), study area, and air pollution. Traffic PM₁₀ and NO₂ were included in regression models. Source-specific air pollution exposure of SAPALDIA participants were modeled by applying Gaussian dispersion models [31]. Additionally considered variables in regression models were self-reports of open windows in the bedroom during the night at any season and home equipped with double-glass windows. Among the studied population, more than 90% of participants claimed having double-glass windows at home and more than 70% of the participants reported sleeping with open windows at night irrespective of season.

2.5. Statistical analysis

Descriptive statistics are presented as mean \pm standard deviation, median, and range. Comparisons between groups were done using unpaired *t* tests or Mann–Whitney tests for independent samples when appropriate. Associations between the distances of individuals' addresses within 100 m or 50 m of main roads and nighttime VLFI were tested using univariate and multivariate logistic regression analyses. Similar models were repeated to test the associations between the distances of individuals' addresses within 100 m or 50 m of main roads and self-reported SDB. Models were adjusted on a stepwise basis first including age, level of education, BMI, smoking status, alcohol intake, hearing deficiency, comorbidities, and study area; then we added the variables of self-reports of sleeping with open windows at any season and having double-glass windows at home. Because spatial correlation between traffic noise and air pollution might exist even though it was weaker in areas with higher traffic density [32], we examined the potential colinearity between the distance of individuals' homes to main roads and individually assigned home outdoor traffic PM₁₀ or NO₂ using Pearson product moment correlation coefficients, including these two air pollution variables in adjusted models.

Interactions of the distance of homes to main roads with a number of independent variables (i.e., age, level of education, hearing deficiency, sleeping with open windows at any season, having double-glass windows at home) were tested by adding multiplicative terms into the models. Because the interactions with sex were significant for the main effects ($P < .0001$), regression models were further stratified by sex.

Sensitivity analyses were performed by restricting models to participants who remained at the same address between baseline and follow-up surveys and by excluding participants who reported depression (less than 10% of the studied population). Additional regression analyses were conducted in all participants stratifying by age (<60 years and ≥ 60 years) and in women stratifying by self-reports of regular use of hormonal replacement therapy (HRT).

All analyses were performed using SPSS version 20.0 (IBM Corp. IBM SPSS Statistics for Windows, Released 2011, Armonk, NY, USA). Two-sided *P* values of .05 and .10 were considered as statistically significant for main effects and interactions, respectively.

3. Results

Analyses comprised 1383 participants (53% women) with approximately 30% living within 100 m of a main road. Table 1 shows the number of participants according to the individual characteristics, stratified by sex. Although there were no relevant differences regarding age and prevalence of chronic morbidities,

male participants had higher self-reported education, significantly higher BMI, higher prevalence of smoking and drinking alcohol, and more reports of auditory deficiency; however, they were less likely to be annoyed by traffic noise than women. In addition, SDB was significantly reported more often by men than women.

Table 2 shows the VLFI results according to the participants' characteristics. The overall mean \pm standard deviation of the VLFI was 5.0 ± 3.0 (median, 4.5; range, 0.1–19.6). Higher VLFI values were found in participants reporting SDB compared to controls (5.8 ± 3.2 vs 4.9 ± 3.0 ; $P = .002$). As shown in Table 2, significantly higher VLFI values were found in men, smokers, participants with higher education, and participants reporting regular alcohol intake and SDB. No significant differences were observed on the VLFI when comparing participants living within 100 or 50 m of main roads to those living further. Stratifying by sex showed that women living close to main roads had significantly higher VLFI compared to those living further (4.5 ± 2.6 vs 4.0 ± 2.4 , respectively; $P = .04$), but this difference was not observed in men (6.0 ± 3.1 vs 5.9 ± 3.3 , respectively; $P = .93$).

Table 3 presents the associations of the distance from home to main roads within 100 m or 50 m and the VLFI stratified by sex. Significant associations between variables were observed in the overall group of participants, with stronger effects when the distance of home to main roads was within 50 m. The results remained consistent after adjustments. Stratified by sex, associations remained significant in women but not in men. Again stronger associations were found when the distance to main roads was less than 50 m, especially in women.

The association between distance of home to main roads and self-reported SDB was marginally significant in men ($P = .05$), but the effect was not seen in adjusted models or in women. No significant interactions were found between the distance from home to main roads and age, level of education, hearing deficiency, or self-reported sleeping with open windows at any season and having double-glass windows at home.

Regular use of HRT was reported by 280 women (38.3%) in our study. The association between distance from home to the main road within 100 m and VLFI was significant in the group of women reporting regular use of HRT (odds ratio [OR], 2.101 [95% confidence interval {CI}, 1.199–3.682]; $P = .01$) but not in their counterparts who did not use this therapy (OR, 1.043 [95% CI, 0.665–1.636]; $P = .855$). Effects were stronger in women under HRT who lived within 50 m of a main road (OR, 2.764 [95% CI, 1.343–5.687]; $P = .006$) and were not significantly relevant in women who did not use HRT (OR, 1.169 [95% CI, 0.669–2.043]; $P = .584$). Stratification by age showed similar and significant effects comparing women ages less than 60 years and 60 years or older who reported regular use of HRT (OR, 3.031 [95% CI, 1.161–7.910]; $P = .023$ vs OR, 3.286 [95% CI, 0.997–10.825]; $P = .051$, respectively). No age effect was observed among nonusers of HRT or men.

Sensitivity analysis restricting the sample to participants who remained in the same address between the SAPALDIA 1 and 2 surveys did not substantially change the direction of results. Associations between the distance to main roads and the VLFI remained significant in women, either living within 100 m (OR, 1.48 [95% CI, 1.02–2.15]; $P = .042$) or 50 m (OR, 1.81 [95% CI, 1.13–2.90]; $P = .013$) of a main road. No significant associations were found between the distance from homes to the main roads and self-reported SDB in participants remaining in the same address between the two surveys. Sensitivity analyses excluding participants reporting depression did not substantially change the associations of VLFI and distance from home to the main road within 100 m (OR, 1.39 [95% CI, 1.04–1.85] [$P = .024$ in all participants]; OR, 1.64 [95% CI, 1.12–2.40] [$P = .011$ in women]; OR, 1.15 [95% CI, 0.74–1.78] [$P = .526$ in men]), or in models using distance from home to the main road within 50 m (OR, 1.62 [95% CI, 1.11–2.35] [$P = .012$ in

Table 1

Characteristics of participants enrolled in our study.

Variables/participants	All N = 1383	Men n = 652	Women n = 731	P value
Age, y	59.8 ± 6.2	59.7 ± 6.0	60.0 ± 6.3	.348
Body mass index, mean ± SD	26.4 ± 4.3	27.0 ± 3.5	25.9 ± 4.8	<.0001
Basic education, n (%)	332 (25)	102 (8)	230 (17)	<.001
Current smokers, n (%)	289 (21)	162 (12)	127 (9)	.001
Regular alcohol intake, n (%)	964 (70)	557 (40)	407 (30)	<.0001
Distance home: main road ≤100 m, n (%)	402 (29)	179 (13)	223 (16)	.190
Distance home: main road ≤50 m, n (%)	206 (15)	85 (6)	121 (9)	.061
Annoyance by traffic noise, n (%)	175 (13)	69 (5)	106 (8)	.028
Hearing deficiency, n (%)	198 (14)	122 (9)	76 (5)	<.0001
Chronic morbidities, n (%)	454 (33)	208 (15)	246 (18)	.501
Self-reported SDB, n (%)	137 (10)	103 (7)	34 (3)	<.0001

Abbreviations: y, years; SD, standard deviation; m, meters; SDB, sleep-disordered breathing.

Chronic morbidities were variables, including arthritis, systemic hypertension, diabetes mellitus, and chronic obstructive pulmonary disease.

SDB defined by self-reports of respiratory pauses during sleep identified by partner or nontreated doctor-diagnosed obstructive sleep apnea syndrome.

Table 2

Median (range) of very low frequency index results according to participants' characteristics.

	VLF results	P value
Men/women	5.4 (0.1–19.6)	<.0001
Body mass index (<25 vs >25)	4.4 (0.1–17.0)	.95
Low/high education	4.4 (0.1–19.6)	.01
Smoking (never smoked/smokers)	4.4 (0.2–17.6)	.05
Regular alcohol intake (no/yes)	3.7 (0.1–19.6)	<.0001
Annoyance by traffic noise at night (no/yes)	4.5 (0.1–19.8)	.06
Hearing deficiency (no/yes)	4.5 (0.1–19.6)	.88
Chronic morbidities (no/yes)	4.6 (0.1–19.6)	.01
Home address ≤100 m of main road (no/yes)	4.4 (0.1–19.6)	.31
Home address ≤50 m of main road (no/yes)	4.4 (0.1–19.6)	.23
Self-reported SDB (no/yes)	4.4 (0.1–19.6)	.001

Abbreviations: VLF, very low frequency index (for details, see Methods); m, meters; SDB, sleep-disordered breathing.

Chronic morbidities were variable combining diabetes mellitus, arterial hypertension, arthritis, and chronic respiratory disorders.

SDB defined by self-reports of respiratory pauses during sleep identified by partner or nontreated doctor-diagnosed obstructive sleep apnea syndrome.

Table 3

Association of the distance from home to main roads within 100 m or within 50 m and nighttime very low frequency index stratified by sex.

Model covariates		Distance ≤100 m n = 402		Distance ≤50 m n = 206	
		OR (95% CI)	P value	OR (95% CI)	P value
All (N = 1383)	Unadjusted	1.225 (0.968–1.552)	.091	1.385 (1.020–1.880)	.037
	Model 1	1.311 (1.009–1.704)	.043	1.477 (1.053–2.071)	.024
	Model 2	1.297 (0.996–1.687)	.053	1.474 (1.049–2.070)	.025
	Model 3	1.346 (0.999–1.814)	.051	1.657 (1.122–2.449)	.011
Men (n = 652)	Unadjusted	1.178 (0.802–1.731)	.404	1.301 (0.770–2.198)	.326
	Model 1	1.185 (0.788–1.783)	.415	1.327 (0.762–2.310)	.317
	Model 2	1.185 (0.786–1.786)	.418	1.347 (0.771–2.354)	.295
	Model 3	1.161 (0.731–1.843)	.527	1.367 (0.725–2.578)	.334
Women (n = 731)	Unadjusted	1.399 (1.019–1.921)	.038	1.699 (1.147–2.517)	.008
	Model 1	1.417 (1.006–1.995)	.046	1.597 (1.043–2.445)	.031
	Model 2	1.389 (0.985–1.959)	.061	1.575 (1.026–2.418)	.038
	Model 3	1.536 (1.034–2.280)	.033	1.939 (1.178–3.194)	.009

Abbreviations: OR, odds ratio; CI, confidence interval.

Model 1: adjusted for age, level of education, body mass index, smoking status, alcohol intake, hearing deficiency, comorbidities, and study area.

Model 2: model 1 plus self-reports of sleeping with open windows during any season and having double-glass windows at home.

Model 3: model 2 plus PM₁₀ and NO₂.

all participants]; OR, 2.01 [95% CI, 1.24–3.27] [$P = .005$ in women]; OR, 1.26 [95% CI, 0.70–2.28] [$P = .442$ in men]). Results were again consistently stronger in models with distance from home to main roads within 50 m.

Potential collinearity of air pollution effects and the effects determined by traffic noise were tested by assessing correlations between measured air pollutants and the distance from individuals' home addresses to main roads. Weak and negative correlations were found for total PM₁₀ ($r = -0.062$; $r^2 = 0.004$ [$P = .031$]), traffic

PM₁₀ ($r = -0.176$; $r^2 = 0.031$ [$P < .0001$]), and NO₂ ($r = -0.226$; $r^2 = 0.051$ [$P < .0001$]).

4. Discussion

Findings of our study showed that measurements of nighttime VLF, a marker of sleep fragmentation, were significantly increased in participants living in proximity to a main road, and the effects were sex dependent. Associations were significant in women,

particularly in those living less than 50 m of a main road, but the results were not significant in men. Furthermore, the effect in women was associated with regular use of HRT. No relevant associations were observed between the distance from home to the main roads and SDB in both men and women. To our knowledge, our study is the first epidemiologic evidence reporting the effects of living in close proximity to busy roads on sleep fragmentation.

Several studies have reported on the detrimental effects of nocturnal exposure to traffic noise emphasizing changes in the sleep structure; alteration of the circadian rhythm; release of stress hormones; and development or aggravation of cardiovascular diseases, particularly systemic arterial hypertension and myocardial ischemia [5,7,17,33–35]. Pathways underlying these biologic effects may relate to direct autonomic heart rate responses triggered by noise during sleep or they may be mediated by arousals; the latter may consequently activate the sympathetic autonomic nervous system stimulating afferent receptors located in the respiratory tract to increase upper airway resistance and inspiratory muscle pressure, leading to intermittent airway obstruction and hypoxemia.

Most of the literature regarding the health effects of noise has focused on cardiovascular disorders, notably arterial systemic hypertension and coronary heart disease. Few studies have examined the impact of noise on autonomic arousals and SDB, conditions which may precede the onset of adverse cardiovascular events. Early experimental work demonstrated that noise-induced sleep fragmentation increased upper airway collapsibility and SDB [11]. Furthermore, examining healthy young volunteers, Griefahn et al. [18] found heart rate increases to be correlated with autonomic arousals and traffic noise stimuli. Our results suggesting increased sleep fragmentation in a large population-based sample confirm these earlier reports derived from laboratory measurements conducted in a restricted number of participants. Moreover, we were able to expand these findings by demonstrating a link between different levels of exposure to traffic noise at night and sympathetic nervous system activity and further showing sex differences in the effects of noise.

In our study, women reported being more disturbed than men by noise and showed a higher risk for sleep fragmentation when living in close proximity to busy roads. In contrast, SDB was not significantly associated with living in proximity to main roads, despite the fact that reports of SDB were 2-fold higher in men than in women in our study. These differing results might indicate different physiopathologic mechanisms in the effects of noise on sleep fragmentation and SDB, the former being a warning sign of noise-related disturbance; however, these physiopathologic mechanisms most likely are not a precursor of SDB. The overall small number of participants who reported having SDB in our cohort and the potential bias misclassification incurred by self-reports could have played a role in the observed negative effects.

Previous studies have reported on the increased sensitivity of women to noise and to other environmental stressors such as smoking [9,36]. Susceptibility to noise changes across lifespan, achieving highest levels around menopausal age [37]. Because the quality of sleep decreases in women after menopause, symptoms of repeated awakenings and less effective sleep frequently are reported. Further, they have been linked to hormonal changes during this life period. Noise-disturbed sleep has been shown to alter neuroendocrine regulation and disrupt physiologic cortisol levels, a stress hormone in menopausal women [33,35]. This disruption could lead to an increased burden of menopausal symptoms, which may consequently prompt more frequent use of HRT. It is possible that women under HRT in our study, who were in turn more susceptible to hormonal changes, also could present more vulnerable to the effects of noise. On the other hand, fluctuating estrogens and progesterone during menopause have been reported

to affect sleep architecture and increase the risk for sleep fragmentation and SDB [38]. However, estrogen replacement therapy has shown contradictory results in reverting these sleep disturbances [39]. In our study, HRT did not contribute to the attenuation of sleep fragmentation.

Strengths of our study include the large population-based sample with a 10-year follow-up, the systematic individuals' examinations during the baseline and the follow-up surveys, the standardized ECG recordings, the longitudinal repertory of individuals' addresses, and the collection of reproductive characteristics allowing us to identify vulnerable women. However, our study also has several limitations. First, we acknowledge the lack of a gold standard for the detection of sleep fragmentation. However, evidence gathered by Basner et al. [40] comparing single-channel ECG tracings and classic polysomnographic electroencephalography monitoring in participants exposed to noise during sleep showed that cardiac activations identified by the two means were similar for the detection of arousals. Although the majority of studies that observed the association between sympathetic activity and sleep fragmentation have been conducted in patients with sleep disorders, validation of the VLF as a marker of sleep fragmentation has been previously published in a large population-based cohort of elderly individuals [24]. In addition, sympathetic arousals during sleep were associated with higher systolic blood pressure in healthy elderly individuals [41]. Second, the absence of realtime records of nocturnal traffic-related noise intensity measured in decibels close to participants' sleeping rooms prevented a more accurate estimation of the magnitude of noise during the nighttime. Distance from homes to main roads has been shown to be an adequate proxy for the evaluation of air pollution effects on participants living close to busy roads [29], but we cannot ensure that the same is valid for the evaluation of noise. In fact, the attempts to reveal the interaction between effects of noise and air pollution on cardiovascular health led to the conclusion that one may need an estimate of nighttime noise exposure indoors in the bedroom [42,43]. Without considering adaptive noise related to coping behavior, there may be biases toward null findings and this bias also may be present for the association between noise exposure and sleep. Finally, self-reported SDB might have induced misclassification. To reduce this bias, we restricted the definition to self-reported SDB confirmed by a doctor and to respiratory pauses identified by the partner. Although it seems unlikely that participants would misinterpret these objective questions, we cannot avoid misunderstandings.

5. Conclusions

Findings of our study showed an approximate 60% higher risk for sleep fragmentation in elderly women living near main roads compared to those living further. Furthermore, we found evidence that these effects were 2-fold higher in women under HRT, underscoring the higher vulnerability of this subset of the population.

Funding sources

SAPALDIA is supported by the Swiss National Science Foundation (grants no 33CSCO-108796, 3247BO-104283, 3247BO-104288, 3247BO-104284, 3247-065896, 3100-059302, 3200-052720, 3200-042532, 4026-028099), the Federal Office for Forest, Environment and Landscape, the Federal Office of Public Health, the Federal Office of Roads and Transport, the canton's government of Aargau, Basel-Stadt, Basel-Land, Geneva, Luzern, Ticino, Valais, and Zurich, the Swiss Lung League, the canton's Lung League of Basel Stadt/Basel Landschaft, Geneva, Ticino, Valais and Zurich, SUVA, Freiwillige Akademische Gesellschaft, UBS Wealth Founda-

tion, Talecris Biotherapeutics GmbH, Abbott Diagnostics, European Commission 018996 (GABRIEL), Wellcome Trust WT 084703MA. The authors are particularly grateful to the Lung League of Valais for supporting this work. None of the funders had any role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: <http://dx.doi.org/10.1016/j.sleep.2013.10.014>.

Acknowledgments

SAPALDIA study directorate: T. Rochat (p), J.M. Gaspoz (c), N. Kunzli (e/exp), N.M. Probst Hensch (e/g), C. Schindler (s).

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The study could not have been done without the help of the study participants, the technical and administrative support and the medical teams and fieldworkers at the local study sites.

Local fieldworkers: Aarau: S. Brun, G. Giger, M. Sperisen, M. Stahel, Basel: C. Burli, C. Dahler, N. Oertli, I. Harreh, F. Karrer, G. Novicic, N. Wyttinbacher, Davos: A. Saner, P. Senn, R. Winzeler, Geneva: F. Bonfils, B. Blicharz, C. Landolt, J. Rochat, Lugano: S. Boccia, E. Gehrig, M.T. Mandia, G. Solari, B. Viscardi, Montana: A.P. Bieri, C. Darioly, M. Maire, Payerne: F. Ding, P. Danieli, A. Vonnez, Wald: D. Bodmer, E. Hochstrasser, R. Kunz, C. Meier, J. Rakic, U. Schafroth, A. Walder.

Administrative staff: C. Gabriel, R. Gutknecht.

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